# UNITED STATES DISTRICT COURT DISTRICT COURT OF NEVADA

LaKISHA NEAL-LOMAX, JOSHUA WILLIAM LOMAX, ALIAYA TIERRAEE LOMAX, JUANITA CARR, as parent and guardian of INIQUE ALAZYA LOMAX, and JOYCE CHARLESTON, individually, and as Special Administrator of the Estate of WILLIAM D. LOMAX, JR.,

Plaintiffs,

VS.

Case No.CV-S-05-01464-PMP-RJJ

LAS VEGAS METROPOLITAN POLICE DEPARTMENT; OFFICER REGGIE RADER, in his individual and official capacity; SHERIFF BILL YOUNG, in his official capacity; TASER INTERNATIONAL, INC., an Arizona Corporation; TASER INTERNATIONAL, INC., a Delaware Foreign Corporation; DOES I through X; DOES XI through XX; and ROE CORPORATIONS XXI Through XXX, inclusive,

Defendants.

Expert Report: Mark Kroll, PhD, FACC, FHRS Box 23 Crystal Bay. MN 55323 Phone: 805-428-1838

Pursuant to Fed. R. Civ. P. 26(a)(2), I, Mark Kroll, hereby submit my report that contains a complete statement of all opinions to be expressed and the bases and reasons therefore; the data and other information I considered in forming the opinions; the exhibits or list of references I used as a summary of or support for the opinions; my qualifications, including a list of all publications authored within the preceding ten years; the compensation to be paid for the study and testimony; and a listing of any other cases in which I have testified as an expert at trial or by deposition within the preceding four years.

MARK W. KROLL, PHD, FACC

April 19, 2007

Date

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## INTRODUCTION

## **Opinion Methodology**

The following opinions were developed using the disciplines of electrical engineering, biomedical engineering, cardiovascular physiology, scientific methods, mathematics, statistics, and physics.

### **Scientific Method**

I have invested most of my adult life researching and developing electrical devices to diagnose and treat disease. My primary scientific specialty is the effect of shocks on the human body. I am proud that this investment has resulted in every ICD (Implantable Cardioverter Defibrillator) made anywhere in the world licensing some of my patented improvements.

Briefly, I research, lecture, and publish on electric shock and their effects on the human body. I have lectured throughout Europe and Asia as well as at the major universities and medical centers of the United States on this topic. With 240 issued United States patents and hundreds of pending and international patents, I currently hold the most patents on electrical medical devices of anyone in the world. I also currently hold the most cardiac device patents.

I am an author on about 150 abstracts, papers, book chapters, and invited presentations and the co-editor of the book, "Implantable-Cardioverter Defibrillator Therapy" published in 1996 by Kluwer of Boston.

I am prepared to speak about various scientific research methodologies that have been used to study the safety of the TASER Electronic Control Devices (ECDs or devices). These include mathematical analyses, computer models, animal studies, and human studies.

## **Compensation and Previous Cases**

My fees for the preparation of this report are \$200 per hour. I have invested about 30 hours in this report and will thus invoice approximately \$6,000. My fees for testimony are \$350 per hour. I have not testified as an expert at trial or by deposition within the preceding four years except in the patent case: Parker v Cardiac Science.

## **Appendices**

This report includes the following general appendices:

- 1. Pre-incident opinions
- 2. Educational exhibits
- 3. Curriculum vitae which includes all publications over the last 10 years

Also, each graphic in this report or any of its references, as well as any documents referenced or cited, as well as any compilation of documents are to be considered exhibits to this report and will likely be utilized as exhibits at deposition and/or trial.

Also included are the following case-specific appendices:

- A. Case Specific Documents Reviewed
- B. Summary of Facts
- C. Incident Summary

I also reviewed the Plaintiff's expert reports of Drs. Bush, Strote, Woodward, and Rhodes.

#### **CONCLUSIONS:**

Based on my review of the documentation listed above, as well as my professional education, experience and background, it is my opinion, to a reasonable degree of medical and scientific probability (certainty), that the use of the TASER Electronic Control Device did not cause or contribute to Mr. Lomax's death. It is also my opinion, to a reasonable degree of medical probability (certainty), that Mr. Lomax's death was caused by excited delirium leading to metabolic acidosis.<sup>1-17</sup>

Mr. Lomax's case reads almost like a textbook example of excited delirium with calculated odds of 52,596 to 1 in favor of excited delirium.

#### Calculations for Odds of Excited Delirium:

As shown below, there are 18 total items on the checklist for an excited delirium with metabolic acidosis death. Mr. Lomax satisfied 15 out of the 16 for which we have data.

Criterion Citat	OII	ints
1. Agitation He was	just tense. Officer Herrera.	1
speech cord	s.	1
Clothing removal Kept	lifting his shirt. Off. Herrera.	1
thou (Wie	gh he asked for medical assistance. rman Depo.)	1
Corr	ell.	1
	pht 4-5 officers for over 10 minutes. ER X26 records, multiple witnesses.	1
7. Constant motion or hyperactivity Wall	king in circles. Off. Herrera.	1
	ER X26 "really didn't do anything." medic Pearson.	1
	ed for medical help and then attacked orities.	1
ion included and a second a second and a second a second and a second a second and a second and a second and a second and	aine abuse admitted to Dr. Hindle on ious emergency.	1
11. Breaking of shiny objects such as glass and mirrors	e noted	0
	n and not screaming as put into am- nce. Off. Rader	1
	foundly acidotic on arrival," Dr. Mul- ndani. Records say pH < 6.5.	1
14. Rhabdomyolysis or kidney damage (if suspect is resuscitated)	oital notation by Dr. Tiu.	1
15. Presenting rhythm of PEA (pulse- less electrical activity) or asystole	stole noted by Paramedic Ritz	1
16. Hyperthermia Hos	pital temperatures of 100-103° F.	1
17. Positive Mash test for dopamine transporter assay in brain test.	Toda Oxaminor ramou to do mio miori	?
- · · · · · · · · · · · · · · · · · · ·	ical examiner failed to do a hair test.	?

Mr. Lomax had 15 of the 16 signs of excited delirium for which data was available. A person in a typical emergency responder situation would have at most 3 of these signs. I performed a chi-square statistical analysis on these ratios and found a Chi-square statistic of 18.286. This equates to a probability of p = .000019 which inverts to an odds ratio of 52,596.

The excited delirium of Mr. Lomax may have been triggered by his past cocaine use, his chronic PCP abuse, and/or by his last PCP high. As Plaintiff's expert Dr. Bush has pointed out, "PCP was developed in the 1950s for use as an anesthetic, but was later abandoned due to its tendency to cause postoperative delirium and hallucinations." PCP is very well known for causing psychosis and delirium. <sup>18-41</sup> In fact one of the few controlled human studies found an ability to cause prolonged toxic psychosis. <sup>42</sup> Ironically, ECT (electroconvulsive therapy) or "shock" therapy has been used to treat the chronic effects of PCP psychosis. <sup>20,26,32</sup>

## TASER X26 Did Not Interfere with Mr. Lomax's Breathing

Human studies with electrodes across the whole chest show that the TASER X26 does not interfere with breathing.<sup>43</sup> The testimony of Officer Radar is that the X26 was applied in drive stun mode to the right side of the back of Mr. Lomax's neck. It is possible, although not based upon scientific or medical evidence that, if the positioning was just perfect then this might have come close to the right phrenic nerve and briefly affected a breath of two.

Even if this happened it is completely irrelevant to the demise of Mr. Lomax for the following reasons:

- 1. The application would have promoted an inspiration, as opposed to respiratory compromise or arrest which might very well have been helpful.
- 2. The right phrenic nerve only drives the right side of the diaphragm and thus the maximum influence was limited to half of the breathing ability.
- 3. There were 7 TASER X26 applications over a period of 10 minutes with an average of 4.4 seconds each. Even if the TASER X26 completely halted his breathing (which it clearly did not) then the effect would be equivalent to holding ones breath for 4.4 seconds which has zero effect. In fact, as taught in any scuba class, the lungs have a 20 second reserve of oxygen even when they are completely exhaled. In other words, an objective reasonable test would be to ask someone to hold their breath for 4.4 seconds which is trivial. To do this 7 times over a 10 minute period is also trivial.
- 4. PCP is an anesthetic (nerve blocker) and its sodium channel blocking capabilities would interfere with the ability to electrically affect the right phrenic nerve.<sup>44</sup>
- 5. Paramedic Pearson testified that the TASER X26 "really didn't do anything" which suggests that it had no effect on the breathing.

6. Paramedic Ritz monitored the breathing of Mr. Lomax and noted no difference with the drive stun application of the TASER X26.

In conclusion, while it is theoretically possible that the TASER X26 could have had a slight impact on the breathing of Mr. Lomax, this impact would have been too small to be noticed and to small to have any negative pathophysiological effect. In fact, there was an equal chance of a slight positive impact.

## **PLAINTIFF'S EXPERTS' THEORIES**

## Observations of Dr. Bush

Dr. Bush has claimed that the blood levels of PCP in Mr. Lomax were less than that required to cause an automatic death. The blood levels in Mr. Lomax ranged from 28 ng/ml up to 129 ng/ml. Dr. Bush cites to a textbook and website and argues that the minimal lethal dose is 1000 ng/ml. However, the paper (Caplan)<sup>1</sup> cited by Dr. Bush, himself, directly contradicts that level and reports PCP fatalities with blood levels down to 148 ng/ml.<sup>45</sup>

The "automatically lethal" level is really only of academic interest in this sad case as the bizarre and violent behavior of Mr. Lomax is also entirely consistent with a "recreational" level of PCP. These behaviors are well documented to include aggression, paranoia, and bizarre behavior. <sup>25</sup>

Dr. Bush is arguing something that is simply not at issue. No one is saying that Mr. Lomax simply ingested an enormous amount of PCP and then fell over dead. However his toxic level of PCP may have triggered his delirium which led to his death. Dr. Bush even admits that when he states:

Similarly, Caplan, et al. reported only 2 of 37 PCP - related fatalities were due to acute intoxication (Caplan, Y.H., Orloff, K.G., Thompson, B.C., and Fisher, R.S., *Detection of phencyclidine in medical examiners' cases.*, Journal of Analytical Toxicology. 3:47 -52, 1979).

(Actually, Dr. Bush lifted this quote verbatim from the Poklis article<sup>46</sup> without attribution.)

In other words, the vast majority (35/37) of PCP fatalities were not due to an automatically lethal level but rather to the triggered behaviors.<sup>46</sup>

Dr. Bush closes with the following:

CAUSATION/CONCLUSION
Within a reasonable degree of medical certainty/probability, phencyclidine

<sup>&</sup>lt;sup>1</sup> This is 1 of 2 pieces of evidence suggesting that Dr. Bush never actually read the Caplan paper that he relies on.

(PCP) intoxication was not a proximate cause of the death of Mr. Lomax.

Dr. Bush might very well be correct depending on his meaning of "proximate." If he meant less than 10 minutes for "proximate" then this conclusion is certainly true. However, this cannot be stretched to state that the PCP was not the main cause of death as its violent and psychotic sequelae are well known and consistent with Mr. Lomax's last minutes of life. And, this conclusion does not speak to the well-known chronic psychoses often seen with PCP usage. 35,42

In the sad but predictable death of Mt. Lomax, even this does not really matter. Yes, it is clear that PCP can trigger violent and dangerous behavior. And, this may have been what triggered the excited delirium episode of Mr. Lomax. But, his past cocaine use could just as well have been the trigger. Due to the failure of the Medical Examiner to perform either a Mash brain test<sup>47-49</sup> or a hair test,<sup>50-56</sup> this other academic issue will never be resolved.

Very notably, Dr. Bush fails to address the very obvious possibility of excited delirium. One would think that he would have brought excited delirium up to eliminate it as a competing cause of death — had he a good argument for eliminating it.

What is clear is that the death of Mr. Lomax was due to his own choices to take dangerous illegal drugs, coupled with his sister's illegal acquisition and supply of the PCP. His death was not the fault of the police officer, security officers, paramedics, handcuffs, restraint straps, or the electronic control device.

#### Theories of Dr. Strote

Dr. Strote notes that Mr. Lomax had numerous problems predisposing him to cardiac arrest including obesity, PCP use, prolonged struggle against restraint, and fibrosis in his heart muscle. Nevertheless, Dr. Strote feels that the TASER X26 somehow contributed to the metabolic derangement of Mr. Lomax and contributed to his hypersympathetic state.

#### Definitions:

Catecholamines (the "hyper hormones") refer primarily to epinephrine and norepinephrine. The British term for epinephrine is "adrenaline" and hence one occasionally hears lay phrases like, "She was so full of adrenaline that she lifted the car." A "sympathetic response" refers to getting hyper (full of "adrenaline") and not to showing sympathy.

Dr. Strote's speculations are faulty for several concrete reasons:

1. He cites the Jauchem Air Force pig study which found some acidosis (acidic blood) in pigs after 90 seconds of TASER device applications.<sup>57</sup> This is inapplicable to the Lomax case for these reasons:

- a. The Air Force pigs were on an anesthetic TZ (tiletamine-zolazepam) is known for compromising respiration<sup>58</sup> and this would cause acidic blood.<sup>2</sup>
- b. The pigs had TASER barbs completely across their trunk and thus put current throughout the whole chest. Mr. Lomax only had a very local current across the back of his neck.
- c. As noted above, it is unlikely that the TASER X26 had any effect on the breathing of Mr. Lomax. And, if there was any effect then it was completely negligible. Any effect had to be so small that two trained paramedics saw no effect on the breathing.
- d. Human studies show that TASER X26 applications decrease rather than increase acidity.<sup>59</sup> Thus, Dr. Strote's speculation is the opposite of scientific fact.
- e. Also, Dr. Jauchem states in a followup published letter:

It is important to note that our exposure conditions were somewhat extreme compared with those commonly experienced during civilian law-enforcement use of TASER International's Advanced TASER X26. Therefore, it would not be prudent to draw conclusions about such use on the basis of our study alone.

- For his alternative speculation, Dr. Strote theorizes that the TASER X26 "contributed to his hypersympathetic state." In other words, the X26 was painful and this made Mr. Lomax more "hyper." This speculation is unscientific for the following reasons:
  - a. PCP is an anesthetic (nerve blocker) and that is for what it was developed. 31,40,60,61 Thus the signals would not get from the site of electrical contact to the brain.
  - b. PCP is a powerful analgesic (pain killer) and thus Mr. Lomax was not feeling any pain. 60-62
  - c. All witnesses agree the X26 ECD had minimal effect on Mr. Lomax.44
  - d. PCP already raises the norepinephrine levels extremely high and thus it is doubtful that anything can add to that.<sup>63</sup>

<sup>&</sup>lt;sup>2</sup> However, the researchers intentionally chose TZ as it had minimal effects on creatine phosphokinase which is a marker for muscles damage. Thus, they intentionally were exaggerating breathing effects in order to focus on theoretical muscle damage (which was not found).

- e. Exercise also dramatically raises the epinephrine and norepinephrine levels by a factor of 7 to 1 and thus it is doubtful that anything can add to that with the extreme level of exertion that Mr. Lomax was under.<sup>64</sup>
- f. The hormones that carry the "hyper' signal are epinephrine and norepinephrine. For the rhythm that Mr. Lomax had, asystole, epinephrine is standard therapy. (In fact, that was given by the paramedics to restore a heartbeat.) Thus it goes contrary to the facts to argue that additional "hyperness" was harmful to Mr. Lomax. Thus, if Dr. Stroted's speculation were true, it would tend to reduce the risk of the lethal rhythm seen in Mr. Lomax

Dr. Strote then goes on to repeat the above speculations with slightly different wording.

Under the heading "Direct Systemic Effects" he argues:

- 1. The TASER ECD exposures could cause significant acidosis.
  - a. This is based on an incorrect extrapolation from the Air Force pig study which is discussed above.
  - b. Recent human studies have clearly shown that the TASER X26 reduces rather than increase acidosis. To be fair to Dr. Strote, the critical studies may not have appeared at the time of the writing of his report.
- 2. "Tasing [sic] almost certainly increased the level of catecholamines in Mr. Lomax. Pain is a well known cause of a sympathetic response."
  - a. TASER is a registered trademark and thus it is improper to construct a verb from this word.
  - b. Dr. Strote's strong use of the phrase "almost certainly" does not substitute for scientific facts. For the following reasons, one can be sure that the X26 had almost no impact on the "level of catecholamines."
    - i. PCP is an anesthetic and that is for what it was developed. 31,40,60,61
    - ii. PCP is a powerful analgesic and thus Mr. Lomax was not feeling any pain. 60-62
    - iii. All witnesses agree the X26 ECD had minimal effect on Mr. Lomax.44
    - iv. PCP already raises the norepinephrine levels extremely high and thus it is doubtful that anything can add to that.<sup>63</sup>

v. Exercise also dramatically raises the epinephrine and norepinephrine levels by a factor of 7 to 1 and thus it is doubtful that anything can add to that with the extreme level of exercise that Mr. Lomax was under.<sup>64</sup>

A more general criticism of the opinion of Dr. Strote is that it contradicts much of what he has recently published before he became involved in this case.<sup>65</sup>

In his paper, "TASER Use in Restraint-Related Deaths" he made the following statements:

In the abstract:

A diagnosis of excited delirium was given for 75.7% of the cases.

In column 2 of page 447:

With increased deployment of the Taser by law enforcement agencies, numerous injuries and deaths have been temporally associated with Taser use, although no direct link to fatal injury has been made.

In column 2 of page 448:

Demonstrating a causal relationship between Taser injury and subsequent death is difficult.

Most telling is a paragraph in column 1 of page 449 where he lists 7 of the criteria from my checklist for excited delirium:

Importantly, the Taser is primarily used in cases where suspects are unarmed and violently resisting arrest, a situation already known to be associated with inrestraint death from excited delirium. Excited delirium is broadly defined as a state of agitation, excitability, paranoia, aggression, great strength, and numbness to pain, often associated with illegal stimulant use and psychiatric disease. In these cases, stimulant use, agitation-related acidosis, hypoxia, and/or rhabdomyolysis are believed to contribute to sudden death, especially in patients who are at higher risk due to underlying heart disease.

He then goes on to admit that subjects were dying from excited delirium long before the TASER ECD existed in column 1 of page 449:

The underlying conditions associated with excited delirium–related deaths in prior studies were overwhelmingly present in the Taser-related deaths reported here.

Dr. Strote agreed that the TASER ECDs were good tools to be used with these difficult cases (column 1 page 449) when he states:

This is not surprising, because Taser weapons were specifically designed to be

useful before restraint in situations in which suspects are displaying signs of excited delirium.

On column 3 of page 449 he states that these patients already have dangerous conditions (such as hypersympathetic state and acidosis) from the excited delirium and/or their struggle against restraint in general:

EMS management of patients recently subjected to application of a Taser should take into account the likelihood of the physiologic results of excited delirium and/or extensive struggle against restraint: hyperthermia, acidosis, rhabdomyolysis, and a hypersympathetic state.

Finally, in his paper's conclusion he appears to contradict his own expert report by again observing that these subjects were dying long before the TASER ECDs existed:

Our data show that sudden deaths can and do occur after Taser use. A common factor in these deaths is extreme agitation, often in the setting of stimulant drug use and/or preexisting heart disease. This finding is consistent with prior studies of restraint-related fatalities.

The most interesting question is this: Why did Dr. Strote, who clearly was familiar with excited delirium, not discuss it in his expert report? In his paper, he recognized that excited delirium was responsible for the vast majority of restraint-related deaths so it should have crossed his mind.

One would think that Dr. Strote would have brought excited delirium up to eliminate it as a competing cause of death — had he a good argument for eliminating it.

## Report of Dr. Woodard

Dr. Woodard gives a long list of 8 factors leading to the death of Mr. Lomax closing with "taser [sic] administration."

Dr. Woodard then amplifies on this by claiming that the TASER ECD "would result in forced muscular contractions..."

This is simply false. In the drive-stun mode there are essentially no muscular contractions. It is possible that Dr. Woodard confused the barb mode (which does have muscular contractions) with the drive-stun mode (which does not). 66-69

He then makes the incorrect claim that this would have interfered with breathing. This is false for many reasons discussed earlier in this report.

Dr. Woodard then appears to take the role of a "use-of-force" expert when he opines:

Additionally, repetitive tasing[sic] because of response to pain would worsen agitation and resistance.

Dr. Kroll Expert Report

This is a very curious comment and it is both inappropriate and incorrect. First, it is inappropriate as Dr. Woodard is not a "use-of-force" expert. He has no experience in subduing violent subjects in the street and thus is not qualified to suggest which restraint technique a professional law enforcement officer should be using under which circumstances.

Secondly, as discussed above, due to the analgesic and anesthetic properties of PCP, Mr. Lomax was feeling no pain.

Finally, this speculation is contrary to the paramedic observations that the TASER ECD had no effect on Mr. Lomax.

## Report of Dr. Rhodes

Dr. Rhodes is completely unqualified to be opining in this case. He has no knowledge of bioelectricity, physiology, or of the heart. He offers numerous bizarre and completely false speculations which are inimical to the pursuit of the truth in this case. Fortunately, these speculations are so bizarre that they make his lack of qualifications very obvious to anyone with knowledge in this area. Were we not dealing with the tragic loss of life, these comments could be rather risible.

Some of Dr. Rhodes more outrageous claims are:

- 1. The TASER ECD made Mr. Lomax go blind.
- 2. The ECD application damaged "the" phrenic nerve.
- 3. A TASER ECD can do serious or permanent damage to nerves if applied near the cervical spine.

Some of the evidence exposing Dr. Rhodes lack of qualifications in this area include his mischaracterization of control of the left and right diaphragm by the phrenic nerves. To summarize briefly, the body has a right hemi-diaphragm that is controlled by the right phrenic nerve and a left hemi-diaphragm which is controlled by the left phrenic nerve. Apparently, in his cursory search for this report, Dr. Rhodes did not understand that there are 2 phrenic nerves and 2 diaphragm muscles. Consider his comments:

While not mentioned in the autopsy, damage to the Phrenic nerve from repeated Taser shocks is consistent with the observed facts in this case: This nerve is in the general area where the shocks were applied, Mr. Lomax was indeed first observed to be in respiratory arrest.

The multiple Taser shocks to the back of his neck, more likely than not acting on the Phrenic nerve, then caused Mr. Lomax to become asphyxiated and go into respiratory arrest. The diaphragm is controlled by the phrenic nerve.

To see how far off his speculation is we can construct an analogy using the ears in place of the hemi-diaphragms. This allows us to see how silly this sounds to a specialist in the area.

The ear is controlled by the acoustic nerve. Damage to acoustic nerve from repeated Taser shocks is consistent with the observed facts in this case: This nerve is in the general area where the shocks were applied, and Mr. Lomax was indeed observed to go deaf.

The issue of the 2 phrenic nerves is important for 2 reasons: First it shows that Dr. Rhodes has no knowledge of bioelectricity and physiology. Even a child knows that a human typically has 2 eyes, ears, arms, and legs as they are visible. With a minimal level of knowledge in bioelectricity, pulmonology, or physiology one would know that there are 2 phrenic nerves and 2 hemi-diaphragms. Secondly, it is material as the loss of one phrenic nerve would no more put someone in respiratory arrest than would the loss of one ear or one eye make someone deaf or blind.

Dr. Rhodes has a PhD in Applied Plasma Physics. His CV shows that he has had interesting and important jobs working on nuclear power and nuclear weapons. He has never worked in the area of bioelectricity, physiology, pulmonology, or cardiology.

Dr. Rhodes has 6 issued US patents. None have anything to do with bioelectricity, physiology, pulmonology, or cardiology.

7,083,985	Coplanar waveguide biosensor for detecting molecular or cellular events
6,627,461	Method and apparatus for detection of molecular events using temperature control of detection environment
6,605,901	Apparatus and method for electrical insulation in plasma discharge systems
5,410,425	Magnetron cathodes in plasma electrode pockels cells
4,731,037	Water survival kit (personal life-raft)
4,719,540	Unitized mounting device for mounting electrical components of a luminaire

Dr. Rhodes has 17 published papers essentially all dealing with plasmas, lasers, and microwaves. None have anything to do with bioelectricity, physiology, pulmonology, or cardiology.

We can now better understand why Dr. Rhodes may have made some of the following claims:

 The TASER ECD blinded Mr. Lomax after the first shock. In fact, full ECD discharges with a barb implanted fully in the eye have not done permanent damage to the vision except for a tiny pinhole from the mechanical presence of the barb. 70,71

- 2. "The evidence shows that his heart was less damaged than his respiratory system because his heartbeat was restored at the hospital with resuscitation procedures but not his respiration." This comment shows a complete lack of understanding of physiology, pulmonology, and cardiology. Neither the heart or the respiratory system need to have permanent "mechanical" damage in order to be arrested. In both cases, the respiratory and cardiac arrest were probably caused by severe acidosis as opined by some of the plaintiff experts.
- 3. "I note that the mechanism for delayed cardiac arrest after electric shock is presently unexplained." It is not clear what the point of this comment was. But it does again show the lack of understanding of basic bioelectricity and cardiology. As discussed in my Appendix 1, the electrical induction of cardiac arrest takes 1-5 seconds and is very well understood from bout 1,000,000 human tests.<sup>72</sup> There is no such thing as a delayed cardiac arrest from electric shock.<sup>73</sup> Simply because this is not understood by Mr. Rhodes does not make it "presently unexplained."
- 4. "It is my opinion is that the Taser current pulses can and do damage nerve cells yet this subject is not addressed by Taser International or its experts." This completely false and ignorant speculation is soundly contradicted by 700,000 human uses with no nerve damage as discussed in my Appendix 1. Also careful computer modeling shows the impossibility of nerve damage. 66-68,74
- 5. "However, it is not clear whether Taser International has carefully considered the possibility of serious (if not permanent) damage to nerves especially in the case of repeated shocks applied near the cervical spine." This is possibly the silliest of all of the irresponsible and ignorant speculations of Dr. Rhodes. It clearly shows how little he knows about these devices. Over 250,000 law enforcement officers have received training hits from TASER ECDS right on the back with the barbs digging in deeply over or near the spine. There has not been a *single* complaint of nerve damage. If Dr. Rhodes is allowed to continue as an expert in this case in spite of his obvious lack of qualifications I intend to play videos of my sons taking full strength TASER ECD applications over their spines.
- 6. "TASER International's training material puts forth the notion that that after receiving a Taser shock, one gets up after the shock cycle ends and its as if nothing ever happened. However, it is known that the Taser promotes traumatic biophysical and biochemical effects that last many minutes after the shock" Dr. Rhodes makes this inflammatory accusation against the Taser Intl company yet provides no scientific reference and, in fact, not a scintilla of evidence. Of course he cannot, as the statement is completely false and contradicted by over 250,000 training uses. If Dr. Rhodes is allowed to continue as an expert in this case in spite of his obvious lack of qualifications I intend to submit videos of 1000s of

police offices taking full strength TASER ECD applications over their spines — and then getting up after the shock cycle ends as if nothing ever happened.

The fact that Dr. Rhodes cannot and will not provide scientific evidence of his wild accusation demonstrates that he is not fit to be an expert witness in this proceeding. The fact that he is willing to speculate wildly and support his speculations with nothing more than the innuendo, "it is known," shows that he is not qualified to be an expert witness in this important case.

In addition, the fact that Dr. Rhodes would not or could not list a single scientific reference regarding Electronic Control Devices is further evidence that his report cannot be taken seriously.

Finally, I note that Dr. Rhodes lists expert testimony in a "stun gun" criminal case. That does not provide any evidence of his qualifications in this case. In fact, based on his report in this case, one could draw the conclusion — depending on the specifics of he "stun-gun" case — that he might not have been qualified in that case either.

### References:

- 1. Allam S, Noble JS. Cocaine-excited delirium and severe acidosis. Anaesthesia 2001;56:385-6.
- 2. Brice JH, Pirrallo RG, Racht E, Zachariah BS, Krohmer J. Management of the violent patient. *Prehosp Emerg Care* 2003;7:48-55.
- 3. Sztajnkrycer MD, Baez AA. Cocaine, excited delirium and sudden unexpected death. *Emerg Med Serv* 2005;34:77-81.
- 4. Stratton SJ, Rogers C, Brickett K, Gruzinski G. Factors associated with sudden death of individuals requiring restraint for excited delirium. Am J Emerg Med 2001;19:187-91.
- 5. Morrison A, Sadler D. Death of a psychiatric patient during physical restraint. Excited delirium--a case report. *Med Sci Law* 2001;41:46-50.
- 6. Ruttenber AJ, McAnally HB, Wetli CV. Cocaine-associated rhabdomyolysis and excited delirium: different stages of the same syndrome. Am J Forensic Med Pathol 1999;20:120-7.
- 7. Ross DL. Factors associated with excited delirium deaths in police custody. *Mod Pathol* 1998;11:1127-37.
- 8. Pollanen MS, Chiasson DA, Cairns JT, Young JG. Unexpected death related to restraint for excited delirium: a retrospective study of deaths in police custody and in the community. *Cmaj* 1998;158:1603-7.
- 9. Ruttenber AJ, Lawler-Heavner J, Yin M, Wetli CV, Hearn WL, Mash DC. Fatal excited delirium following cocaine use: epidemiologic findings provide new evidence for mechanisms of cocaine toxicity. *J Forensic Sci* 1997;42:25-31.
- 10. O'Halloran RL, Lewman LV. Restraint asphyxiation in excited delirium. Am J Forensic Med Pathol 1993;14:289-95.
- 11. DiMaio T, VJM D. Excited delirium syndrome cause of death and prevention. Boca Raton: Taylor & Francis, 2006.
- 12. Paquette M. Excited delirium: does it exist? Perspect Psychiatr Care 2003;39:93-4.
- 13. Blaho K, Winbery S, Park L, Logan B, Karch SB, Barker LA. Cocaine metabolism in hyperthermic patients with excited delirium. *J Clin Forensic Med* 2000;7:71-6.
- 14. Park KS, Korn CS, Henderson SO. Agitated delirium and sudden death: two case reports. *Prehosp Emerg Care* 2001;5:214-6.
- 15. Karch SB, Wetli CV. Agitated delirium versus positional asphyxia. *Ann Emerg Med* 1995;26:760-1.
- 16. Wetli CV, Mash D, Karch SB. Cocaine-associated agitated delirium and the neuroleptic malignant syndrome. Am J Emerg Med 1996;14:425-8.
- 17. Mirchandani HG, Rorke LB, Sekula-Perlman A, Hood IC. Cocaine-induced agitated delirium, forceful struggle, and minor head injury. A further definition of sudden death during restraint. Am J Forensic Med Pathol 1994;15:95-9.
- 18. Allen RM, Young SJ. Phencyclidine-induced psychosis. Am J Psychiatry 1978;135:1081-4.
- 19. Carls KA, Ruehter VL. An evaluation of phencyclidine (PCP) psychosis: a retrospective analysis at a state facility. Am J Drug Alcohol Abuse 2006;32:673-8.
- 20. Dinwiddie SH, Drevets WC, Smith DR. Treatment of Phencyclidine-Associated Psychosis with ECT. *Convuls Ther* 1988;4:230-235.
- 21. Fauman BJ, Fauman MA. Phencyclidine psychosis. IMJ Ill Med J 1978;154:324-5.
- 22. Gabbert JF, Giannini AJ. Clinical Therapeutic Conference: dopaminergic/serotonergic actions of phencyclidine as a model for schizophreniform psychosis. *Am J Ther* 1997;4:159-63.
- 23. Giannini AJ, Eighan MS, Loiselle RH, Giannini MC. Comparison of haloperidol and chlor-promazine in the treatment of phencyclidine psychosis. *J Clin Pharmacol* 1984;24:202-4.

- 24. Giannini AJ, Nageotte C, Loiselle RH, Malone DA, Price WA. Comparison of chlorpromazine, haloperidol and pimozide in the treatment of phencyclidine psychosis: DA-2 receptor specificity. *J Toxicol Clin Toxicol* 1984;22:573-9.
- 25. Griffin PT, Garey RE, Daul GC, Goethe JW. Sex and race differences in psychiatric symptomatology in phencyclidine psychosis. *Psychol Rep* 1983;52:263-6.
- 26. Grover D, Yeragani VK, Keshavan MS. Improvement of phencyclidine-associated psychosis with ECT. *J Clin Psychiatry* 1986;47:477-8.
- 27. Jacob MS, Carlen PL, Marshman JA, Sellers EM. Phencyclidine ingestion: drug abuse and psychosis. *Int J Addict* 1981;16:749-58.
- 28. Luisada PV. The phencyclidine psychosis: phenomenology and treatment. NIDA Res Monogr 1978:241-53.
- 29. Luisada PV, Brown BI. Clinical management of the phencyclidine psychosis. Clin Toxicol 1976;9:539-45.
- 30. Marwaha J. Candidate mechanisms underlying phencyclidine-induced psychosis: an electrophysiological behavioral, and biochemical study. *Biol Psychiatry* 1982;17:155-98.
- 31. Rainey JM, Jr., Crowder MK. Prolonged psychosis attributed to phencyclidine: report of three cases. Am J Psychiatry 1975;132:1076-8.
- 32. Rosen AM, Mukherjee S, Shinbach K. The efficacy of ECT in phencyclidine-induced psychosis. J Clin Psychiatry 1984;45:220-2.
- 33. Russ C, Wong D. Diagnosis and treatment of the phencyclidine psychosis: clinical considerations. *J Psychedelic Drugs* 1979;11:277-82.
- 34. Schroeder U, Schroeder H, Darius J, Grecksch G, Sabel BA. Simulation of psychosis by continuous delivery of phencyclidine from controlled-release polymer implants. *Behav Brain Res* 1998;97:59-68.
- 35. Wright HH, Cole EA, Batey SR, Hanna K. Phencyclidine-induced psychosis: eight-year follow-up of ten cases. *South Med J* 1988;81:565-7.
- **36.** Wright HH, Sheth PB, Stasiowski MS. Phencyclidine-induced psychosis. *South Med J* 1980;73:955-6.
- 37. Carlin AS, Grant I, Adams KM, Reed R. Is phencyclidine (PCP) abuse associated with organic mental impairment? Am J Drug Alcohol Abuse 1979;6:273-81.
- 38. Iglesias Lepine ML, Pallas Vilaronga O, Lopez Casanovas MJ, Pedro-Botet J. [Phencyclidine, PCP or "angel dust": a forgotten drug]. *Med Clin (Barc)* 2004;122:276.
- 39. Pearlson GD. Psychiatric and medical syndromes associated with phencyclidine (PCP) abuse. *Johns Hopkins Med J* 1981;148:25-33.
- **40.** Wilson AE, Domino EF. Plasma phencyclidine pharmacokinetics in dog and monkey using a gas chromatography selected ion monitoring assay. *Biomed Mass Spectrom* 1978;5:112-16.
- 41. Yago KB, Pitts FN, Jr., Burgoyne RW, Aniline O, Yago LS, Pitts AF. The urban epidemic of phencyclidine (PCP) use: clinical and laboratory evidence from a public psychiatric hospital emergency service. *J Clin Psychiatry* 1981;42:193-6.
- **42.** Pradhan SN. Phencyclidine (PCP): some human studies. *Neurosci Biobehav Rev* 1984;8:493-501.
- **43.** Ho JD, Dawes DM, Bultman LL, Thacker JL, Skinner LD, Bahr JM, Johnson MA, Miner JR. Respiratory Effect of Prolonged Electrical Weapon Application on Human Volunteers. *Acad Emerg Med* 2007.
- **44.** Allaoua H, Chicheportiche R. Anaesthetic properties of phencyclidine (PCP) and analogues may be related to their interaction with Na+ channels. *Eur J Pharmacol* 1989;163:327-35.

- **45.** Caplan Y, Orloff K, Thompson B, Fisher R. Detection of phencyclidine in medical examiners' cases. *Journal of Analytical Toxicology* 1979;3:47-52.
- 46. Poklis A. Detection of phencyclidine in medical examiners' cases. *Journal of Analytical Toxicology*, 1979;3:47-52.
- 47. Mash DC, Ouyang Q, Pablo J, Basile M, Izenwasser S, Lieberman A, Perrin RJ. Cocaine abusers have an overexpression of alpha-synuclein in dopamine neurons. *J Neurosci* 2003;23:2564-71.
- **48.** Mash DC, Pablo J, Ouyang Q, Hearn WL, Izenwasser S. Dopamine transport function is elevated in cocaine users. *J Neurochem* 2002;81:292-300.
- 49. Mash DC, Staley JK. D3 dopamine and kappa opioid receptor alterations in human brain of cocaine-overdose victims. Ann NY Acad Sci 1999;877:507-22.
- 50. Stephens BG, Jentzen JM, Karch S, Mash DC, Wetli CV. Criteria for the interpretation of cocaine levels in human biological samples and their relation to the cause of death. *Am J Forensic Med Pathol* 2004;25:1-10.
- 51. Berankova K, Habrdova V, Balikova M, Strejc P. Methamphetamine in hair and interpretation of forensic findings in a fatal case. Forensic Sci Int 2005;153:93-7.
- **52.** Bell MD, Rao VJ, Wetli CV, Rodriguez RN. Positional asphyxiation in adults. A series of 30 cases from the Dade and Broward County Florida Medical Examiner Offices from 1982 to 1990. *Am J Forensic Med Pathol* 1992;13:101-7.
- **53.** Kimura H, Mukaida M, Mori A. Detection of stimulants in hair by laser microscopy. *J Anal Toxicol* 1999;23:577-80.
- **54.** Takayama N, Tanaka S, Hayakawa K. Determination of stimulants in a single human hair sample by high-performance liquid chromatographic method with chemiluminescence detection. *Biomed Chromatogr* 1997;11:25-8.
- 55. Kintz P, Cirimele V, Tracqui A, Mangin P. Simultaneous determination of amphetamine, methamphetamine, 3,4-methylenedioxyamphetamine and 3,4-methylenedioxymethamphetamine in human hair by gas chromatography-mass spectrometry. *J Chromatogr B Biomed Appl* 1995;670:162-6.
- **56.** Nagai T, Kamiyama S, Nagai T. Forensic toxicologic analysis of methamphetamine and amphetamine optical isomers by high performance liquid chromatography. *Z Rechtsmed* 1988;101:151-9.
- 57. Jauchem JR, Sherry CJ, Fines DA, Cook MC. Acidosis, lactate, electrolytes, muscle enzymes, and other factors in the blood of Sus scrofa following repeated TASER exposures. Forensic Sci Int 2006;161:20-30.
- 58. Lagutchik MS, Januszkiewicz AJ, Dodd KT, Martin DG. Cardiopulmonary effects of a tile-tamine-zolazepam combination in sheep. Am J Vet Res 1991;52:1441-7.
- **59.** Ho J. Physiologic Effects of Prolonged Conducted Electrical Weapon Discharge on Acidotic Adults. *SAEM* 2007.
- **60.** France CP, Snyder AM, Woods JH. Analgesic effects of phencyclidine-like drugs in rhesus monkeys. *J Pharmacol Exp Ther* 1989;250:197-201.
- 61. Melby EC, Jr., Baker HJ. Phencyclidine for analgesia and anesthesia in simian primates. J Am Vet Med Assoc 1965;147:1068-72.
- **62.** Mallick NP. Phencyclidine analgesia in multiple rib fractures. *Lancet* 1963;1:392-3.
- 63. Contreras P, Monohan J, Lanthorn T, Pullan L, DiMaggio D, Handlemann G, Gray N, ODonohue T. Phencyclidine. Physiological actions, interactions with excitatory amino acids and endogenous ligands.

. Mol Neurobiol. 1987;1(3):191-211.

- 64. Brooks S, Burrin J, Cheetham M, Hall G, Yeo T, Williams C. The responses of the cate-cholamines and beta-endorphin to brief maximal exercise in man. Eur J Appl Physiol Occup Physiol. 1988;57(2):230-234.
- 65. Strote J, Range Hutson H. Taser use in restraint-related deaths. Prehosp Emerg Care 2006;10:447-50.
- 66. Panescu D. Less-than-lethal weapons: Design and Medical Safety of Neuromuscular Incapacitation Devices. *IEEE Eng Med Biol Mag* 2007;26(4).
- 67. Panescu D, Kroll M, Efimov I, Sweeney J. Finite Element Modeling of Electric Field Effects of TASER Devices on Nerve and Muscle IEEE Engineering in Medicine and Biology. New York City, NY, 2006.
- 68. Stratbucker R, Kroll M, McDaniel W, Panescu D. Cardiac Current Density Distribution by Electrical Pulses from TASER devices IEEE Engineering in Medicine and Biology. New York City, NY, 2006.
- 69. Sweeney J. Skeletal muscle response to electrical stimulation. In: Reilly J, ed. Electrical Stimulation and Electropathology New York City: Cambridge University Press, 1992:285-327.
- 70. Chen SL, Richard CK, Murthy RC, Lauer AK. Perforating ocular injury by Taser. Clin Experiment Ophthalmol 2006;34:378-80.
- 71. Ng W, Chehade M. Taser penetrating ocular injury. Am J Ophthalmol 2005;139:713-5.
- 72. Kroll M, Tchou P. Testing of Implantable Defibrillator Functions at Implantation. In: Ellenbogen K, Kay G, Lau C, Wilkoff B, eds. Clinical Cardiac Pacing, Defibrillation and Resynchronization Therapy. Philadelphia: W.B. Saunders Company, 2006:531-557.
- 73. Ideker R, Dosdall D. Can the Direct Cardiac Effects of the Electric Pulses Generated by the TASER X26 Cause Immediate or Delayed Sudden Cardiac Arrest? Am J Forens Med 2007;28.
- 74. Kroll M, Panescu D. Theoretical Considerations Regarding The Safety of Law Enforcement Electronic Control Devices Bioelectromagnetic Society Annual Conference. Cancun, Mexico, 2006.

Appendix 1

# Safety of TASER® Electronic Control Devices

## **Pre-Incident Opinions**

of

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April 17, 2007

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**Pre-Incident Opinions** are those professional opinions that focus on the medical safety of the TASER electronic control devices (ECDs or devices) prior to the instant matter. I reserve the right to amend these opinions based upon additional testimony and/or discovery documents.

## 1. What is Electrocution?

Electrocution is the induction of a cardiac arrest by electrical shock. This is theoretically how a TASER ECD, could kill a criminal suspect. It is an immediate death — on the order of seconds — as electricity does not build up in the body like a poison. 1-9

The electrical induction of ventricular fibrillation (VF) has recently become one of the best-studied causes of death. Paradoxically, this is due to the implantation of lifesaving implantable cardioverter defibrillators (ICDs) about 500 times per day when a cardiac electrophysiologist (EP) will intentionally electrocute a patient. The ICD will then recognize the ensuing VF and deliver a lifesaving shock thus verifying the ICD's proper function.

From this experience of over 1,000,000 such intentional electrocutions, certain facts have been medically and scientifically established beyond any shadow of a doubt:

- 1. VF is either induced or not induced within 1-4 seconds.1
- 2. The cardiac pulse disappears immediately.
- 3. The patient loses consciousness within 5-15 seconds.
- 4. A defibrillation shock—either internal or external—restores a sinus rhythm 99.9% of the time.
- 5. There is no increased risk of a later VF since electrical current does not linger in the body as a poison or drug might.

These facts are appreciated by few newspaper reporters, as can be ascertained by "TASER-related deaths" headlines. Fortunately, the majority of medical examiners do understand these scientific facts as can be seen from the increasing sophistication of autopsies in the same cases. Unfortunately, a few earlier examiners—especially those who failed to consult with an EP—did not share this understanding.

As amazing as it may sound, there are medical examiners even *unaware* of the existence of this important medical specialty. 12

This human experience is entirely consistent with 70 years of animal electrical safety studies and international and Underwriters Laboratories standards.

<sup>&</sup>lt;sup>1</sup> This must not be confused with the more complex process of using elaborate pulse sequences to induce a non-fibrillation tachycardia.

## 2. No Single TASER ECD Pulse Could Stop the Heart

## **Summary**

The TASER ECD cannot stop the heart. 13 While it has brief high currents, just like a strong static electricity shock, the pulses are significantly too short in duration to affect the heart.

The safety of the TASER devices has been verified in numerous human and animal studies. It is doubtful that any law enforcement use-of-force technique or tool has been tested so thoroughly for safety.

## **Safety Margin Calculations**

The best evidence for the cardiac safety of the device lies in the 250,000 voluntary training exposures and 450,000 suspect uses (total of 700,000 human uses) without any credible evidence of a resulting cardiac arrhythmia.

Nevertheless, we can quantify the safety margin of the TASER device outputs by comparing them to published scientific data on the electrical induction of VF (the main cause of cardiac arrest).

There are 2 primary ways to deliver an electrical shock to induce VF. The first is to deliver a single shock exactly during a particularly vulnerable point in the heartbeat cycle. The vulnerable spot is called the "T-wave." See Figure 1. More accurately it is the first half of the T-wave. 14

The suggestion that the TASER device could induce VF (ventricular fibrillation) by shocking into the vulnerable period has been referred to as "Russian Roulette," "Lightning Lottery," or the "Tailspin."

An ACLU report, with some of the cardiologist quotes, might give one the impression that each time a police officer uses the TASER, they are playing a lottery game. And, once in a while, they will hit a lucky (or un-lucky) spot and kill the suspect. They quote Dr. Kathy Glatter as saying, If I hit the heart or create electricity in the wrong time of the (beat) cycle, it could send the whole heart into an electrical tailspin. Dr. Zian Tseng had a more colorful metaphor when he stated, I think they are dangerous...you are shocking someone repeatedly, it becomes a bit like Russian Roulette. At some point, you may hit that vulnerable period."

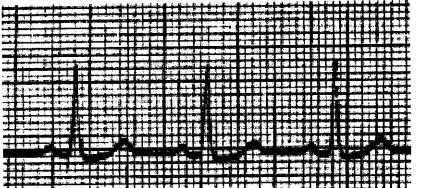


Figure 1. Normal EKG. The tall spike is the R-wave. The rounded positive humps before and after are the P-wave and T-wave respectively.

This T-wave is the "vulnerable" period of the heartbeat as this is when the heart is the most sensitive to an electric shock causing VF (ventricular fibrillation or cardiac arrest). So, Drs. Glatter and Tseng were simply explaining that a strong shock in the T-wave could induce VF. To this extent, the comments are absolutely true. Are these comments relevant to the TASER? Not really. They are somewhat irrelevant for two reasons. The most vulnerable section is the first half of the T-wave and this lasts about 54 milliseconds on average in humans. The TASER X26 weapon puts out 19 pulses per second. That means that the spacing between pulses is about 1/19 second or 52.6 ms. In other words, for the average individual, no vulnerable period escapes the TASER pulsing. This would be an easy lottery to win. A lottery that paid off for every ticket bought.

The second way to deliver a shock to induce VF is to deliver either continuous or nearly continuous current for a longer period of time. For this scenario the critical parameter is the average current which is dealt with in Section 3.

## Safety Margin for a Single Pulse in the Vulnerable Zone

The TASER M26 main phase delivered charge is about 85 microcoulombs ( $\mu$ C) and the TASER X26 delivers about 100  $\mu$ C. It has been known for over a century that the critical parameter of electrical stimulation by short pulses is the electrical charge. This has been more recently confirmed for cardiac stimulation with short duration pulses, such as those from the TASER ECDs.

## **Green Model**

Green investigated the VF induction threshold for canines stimulated along their long axis. <sup>17</sup> He found that the lowest threshold occurred with a 5 millisecond (ms) quarter sine wave and was 2 amperes (A). Integrating, we find a minimum required charge of 6.5 millicoulombs (mC). On the basis of many studies, it is common practice to use a normalization factor of 2 to 1 to go from canine results to human as humans are so much larger than the small dogs typically used in these laboratory experiments. <sup>18</sup> This suggests a human VF threshold of 13 mC. With the TASER X26 charge of 100 μC we can calculate the safety margin as:

For the TASER M26 the safety margin would then be even higher at:

SM = 
$$13 \text{ mC} \div 85 \mu\text{C}$$
  
=  $153:1$ 

## Peleska Model

Peleska studied capacitor discharges into the T-wave and found a minimum required charge of 5 mC to induce VF.<sup>19</sup> This suggests a human threshold of 10 mC.

The safety margin is thus:  $100:1 = 10 \text{ mC} \div 100 \mu\text{C}$  for the X26 and:

118:1 = 10 mC 
$$\div$$
 85 µC for the TASER M26

## **International Safety Standards**

Revisions to the international electrical safety standard IEC-479 are in process which would include scientific measurements directly related to the risk of fibrillation such as the charge in microcoulombs. The proposal refers to 100  $\mu C$  as "disagreeable" and 10,000  $\mu C$  as "ventricular fibrillation likely." Note that this second number is 100 times greater than the TASER ECD charge.

## Conclusion

No pulse from the TASER X26 or the M26 could ever induce VF by affecting the heart during the "sensitive" spot. The charge in the device pulses is too low by factors of at least 100:1. The "Lightning Lottery," "Russian Roulette," and "Tailspin" theories are simply not possible.

## 3. Multiple Pulses from the TASER ECD Cannot Stop the Heart

Since it is clear that no single pulse from a TASER ECD can induce VF, it is important to analyze the impact of the stream of 19 pulses per second (PPS). For the continuous current modeling, we need to calculate the "continuous" current level. With a charge of 100  $\mu$ C delivered 19 times per second we have 1.9 mC per second delivered, which is an average current of 1.9 milliamperes (mA) for the TASER X26. The pulse rate for the TASER M26 varies from 15 to 20 PPS depending on the battery voltage. With the charge of 85  $\mu$ C the average current varies from 1.3-1.7 mA. The more conservative 1.7 mA is so close to the typical 1.9 mA from the TASER X26 that we can simplify calculations by using a conservative 1.9 mA for both devices. (Counting both positive and negative phases of the M26 and X26 gives slightly higher numbers of 3.6 mA and 2.1 mA. In my opinion, the lower numbers are the most appropriate metric for indicating the abil-

ity of the pulses to affect the heart as the polarity changing phases merely offer cancellation effects.)

## Knickerbocker Model

Knickerbocker studied the VF threshold with 20 Hz (hertz or cycles-per-second) AC (alternating current).<sup>21</sup> He found a threshold of 70 mA for canines in the long axis and a 2 second connection. This suggests a human threshold of 140 mA.

This gives a safety margin of 74:1 = 140 mA  $\div$  1.9 mA for the X26 and M26.

## **Beigelmeier Model**

Beigelmeier's model holds that long-term AC (5 seconds or more) requires 1/20<sup>th</sup> of the current to fibrillate as does a single 20 millisecond (ms) pulse (which single pulse would have to be into the T-wave).<sup>4</sup> We can predict the single pulse VF threshold from the Jones and Geddes study which found a ratio of 52:1 for a 10 ms pulse (the widest they investigated). This would give a single 10-ms pulse a VF threshold of 1.4 A = 52 • 27 mA.

Dividing this by Beigelmeier's ratio of 20 gives a predicted AC threshold of 70 mA. Doubling this for humans gives us a human threshold of 140 mA and a safety margin of:

74:1 for the TASER X26 and TASER M26

(which matches the results from the Knickerbocker model)

## **Dalziel Model**

Dalziel performed a meta-analysis of VF thresholds for mammals.<sup>22</sup> This is shown in Figure 2. Conservatively, he included swine which are very easy to fibrillate due to subtle cardiac conduction system differences.<sup>23</sup> The average current required to cause fibrillation is given by the formula:

I = 3.68W + 28.5 mA

Where W is the body weight in kilograms. For pounds the formula is:

I = 1.67W + 28.5 mA

For a typical difficult arrestee of 190 lbs (86 kg) the average current required to fibrillate would be:

I = 346 mA

which gives an average safety margin of 182. However, Dalziel recognized that sensitivities to electrical currents vary and he thus performed a statistical analysis. He plotted the lower limit of sensitivity as the middle line in the figure. Note

that this middle line gives a fibrillating current of over 160 mA for our 190 lb resisting human subject. This gives a safety margin of:

 $84 = 160 \text{ mA} \div 1.9 \text{ mA}$ 

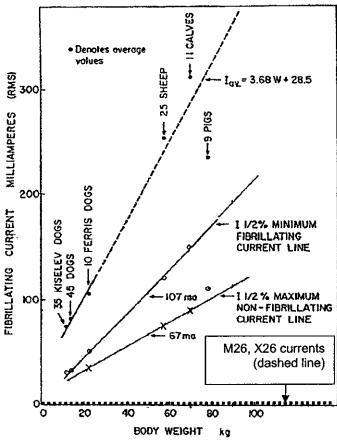


Figure 2. Fibrillating currents as a function of body weight. Note that the ~2 mA ECD currents are barely off of the zero line.

### Conclusion

We have analyzed the risk of the induction of VF from the TASER X26 and M26 ECD with the 2 scenarios known for cardiac risk.

For the risk of delivering a pulse into the vulnerable period of the cardiac cycle we find that the charge required is about 100–130 times that which the TASER X26 delivers. The TASER M26 safety margin was calculated at an even higher range of 118-153:1.

We also analyzed the risk for the delivery of "continuous" current. We found a safety margin of 140:1 by 2 different methodologies for the TASER X26 and TASER M26.

It appears that the TASER device output is incapable of inducing VF in adult human beings. These analyses are based on currents through the trunk. Currents passing from arm to arm have higher safety margins. Other paths such as within a leg have essentially infinite safety margins. With a barb tip practically touching the heart, the margins are lower as will be discussed later.

## 4. Animal Study Results

TASER sponsored a study at the University of Missouri, Columbia, to determine the safety margin of the TASER X26 ECD. This institution has a well founded reputation for leading research in defibrillation going back decades. This study used pigs for the testing as pigs — pound for pound — are the easiest mammals to put into VF (ventricular fibrillation or cardiac arrest) by electricity. Also, the anesthetic used, isoflurane, is known to increase the risk of VF<sup>25</sup> and thus this study was extremely conservative.

Even with the smallest pigs (only 60 pounds) the output of the TASER X26 had to be increased by a factor of 15 to ever cause VF. In fact, a 200-pound pig required over 30 times the X26 output to ever cause VF. This safety margin of 30:1 is far higher than that of many over-the-counter medications. For example, acetaminophen (the active ingredient in many headache and pain medications) has a safety margin of only about 10:1 for the recommended dosage. <sup>26,27</sup>

These ground breaking results were peer-reviewed and published in the leading journal Pacing and Clinical Electrophysiology (PACE). This study was done with careful scientific procedures and set a new standard for use of force weapons research. No one has been able to make a credible criticism of the methodology or principles of this study. And — importantly — no other studies have contradicted the results. On the contrary, other completed studies now in the publication-review process strongly support those results. In fact, the data was validated by a multi-year, multi-million dollar study completed by the United Kingdom government in 2005 when they found an even higher safety margin. <sup>29</sup>

One group failed to induce VF in pigs and was finally forced to surgically open the skin, and move the fat and muscle insulating the heart. Then they poured a conductive gel (which conducted about 8 times better than the removed fat and the muscle against its grain) in until it directly contacted the heart. Only then were they able to induce VF by inserting a barb into the conductive gel within 5/8 inch (17 mm) of the heart.<sup>30</sup> This was a very useful result as it showed the great lengths that one had to go to in order to get VF from a TASER device. Unfortunately, this group perhaps went a bit too far by trying to analogize this extreme experiment to humans even though the thinnest humans have 1.2 inches between their skin and the heart which is filled with highly insulative muscle and fat. They estimated a probability of 0.000 172 of inducing VF with a typical street usage.

The estimate of a 0.000 172 probability is, frankly, dramatically inconsistent with the actual human results of 0 cases of VF in the 450k suspect uses (p = 3.1 • 10-22 by Yates  $\chi^2$ ). Possible reasons for the error include: (1) the use of isoflurane which lowers the VFT (ventricular fibrillation threshold),<sup>25</sup> (2) use of an echocardiographic measurement of skin-to-heart distance instead of the more accurate CT scans, (3) the use of the minimum distance instead of the average over the ventilation cycle, (4) the assumption that the pig VFT is the same as humans when all other mammals require 50% more mass-normalized current for VF than do pigs,<sup>24</sup> and (5) the use of a low resistivity gel to provide a direct current path to the heart.

With a low resistivity gel of only 300  $\Omega$  cm — providing a direct current path to the heart — they were able to induce VF with dart tip-to-heart distances of 17  $\pm$  6.5 mm. Using actual intact pigs Cleveland Clinic researchers inserted TASER CEW barbs to their full depth over the cardiac apex. They achieved tip-to-heart spacings of 16  $\pm$  3.3 mm which is not significantly different from the Wu spacings. However, the Cleveland Clinic *never* induced VF.<sup>31</sup>

The best explanation for these erroneous results (17 mm barb to heart critical spacing) lies with the low and isotropic resistivity gel. There is almost nothing relevant in the chest wall that has such a low resistivity . Fat is about 2500  $\Omega$  cm and the transverse (against the grain) impedance of the skeletal muscle is 2300  $\Omega$  cm.  $^{32}$  The longitudinal resistivity can be as low as 150  $\Omega$  cm but this is not directly relevant in helping currents penetrate perpendicularly into the heart. In fact, MRI studies demonstrate that the majority of current flow in the chest is through the intercostal muscles.  $^{33}$  Also, the difference in fiber orientation between the intercostal muscles and the heart generates a perpendicular current flow making coupling even more difficult.  $^{33}$ 

In summary, replacing the intercostal muscles with a low resistivity gel generated a highly nonphysiological model and most likely explained the misleading results.

In 2006 Nanthakumar et al presented a study of TASER ECDs using 6 pigs averaging 50 kg (110 lb).<sup>34</sup> In the worst-case barb position they achieved ventricular capture but induced no arrhythmias during 94 full strength applications. This is impressive as swine are more sensitive to electrical induction of arrhythmias than are other mammals<sup>24</sup> possibly due to the transmural penetration of the Purkinje fibers.<sup>23</sup>

The researchers then infused epinephrine and delivered 16 ECD applications — again in the worst-case position. They had 1 case of ventricular fibrillation (VF) from these 16 applications. The authors concluded that these devices may have cardiac risks. The first concern lies with the implicit assumption that the rhythm with a police "in-custody death" is VF. ECDs are involved in 30-32% of in-custody deaths. However, studies have not reported a single case in which the presenting rhythm was VF when an ECD was used. A study of 162 consecutive incustody-deaths found that while there was a significant association of impact

weapons with sudden death, the ECDs were never (0/50, p = .001) associated with a sudden collapse. This would seem to eliminate electrically induced VF as the cause of death.

The 1 anecdote, cited in the Nanthakumar paper, of possible electrically induced VF was misreported with material omissions. A violent subject exhibiting all of the signs of excited delirium was briefly subdued with a short ECD discharge. Paramedics were present and found a normal pulse and respiration after the ECD discharge. After a delay of 14 minutes the subject collapsed and probably had an ideoventricular rhythm. After an aggressive therapy of 3 defibrillation shocks along with atropine and epinephrine the subject finally had the VF strip shown in the published anecdote. A total of 23 minutes elapsed between the ECD application and the published VF strip.

The second concern has to do with the timing of the shocks with the epinephrine infusion. The paper does not mention any delay between the time of infusion and the ECD application. While perhaps counterintuitive, epinephrine reduces the VF threshold only for the first few minutes.<sup>39</sup> After that t the VF threshold is increased significantly *above* the baseline. This timing is helpful for the clinical scenario. Typically, an individual exhibits violent agitation with hyperactivity<sup>40</sup> for several minutes and third parties call for help. The police require 5-15 minutes to arrive before they can apply any restraint device. At this point, the adrenergic tone has been elevated for several minutes. An epinephrine infusion just minutes before an ECD application would appear to give results *opposite* of those seen in real life.

Finally, one should draw attention to the results of the Cleveland Clinic study published in the same issue. <sup>41</sup> This study used significantly higher exposures (total shock charge of approximately 2000 5-second weapon discharges per pig vs. about 50 for the Nanthakumar paper) and also evaluated the risk of the induction of ventricular arrhythmias. They found no induction of arrhythmias except at a high multiple of the device output and that cocaine increased this safety margin even farther. That would appear to be more consistent with the clinical results in which no objectively documented VF has occurred in 700,000 police uses of these devices.

## 5. Human Studies Show that the TASER ECD Does Not Affect the Heart

The following study was performed under the auspices of the Hennepin County Medical Center. Adult volunteers (n=66, age  $40.3 \pm 6.8$  years, 65 male, 1 female) had typical cardiovascular histories: 6 hypertension, 6 hypercholesterolemia, 1 each of myocardial infarction and bypass grafting, heart failure, coronary disease, transient ischemic attack, and diabetes with 51 reporting no significant history. Each was shot in the back with standard TASER device barbs

and received the full 5-second application from the law enforcement model TA-SER X26. Each had blood drawn before, immediately after, and at 16 and 24 hours post-exposure. Troponin I, potassium, creatine kinase, lactate, and myoglobin were tested. A 12-lead ECG was recorded in 32 randomly chosen subjects at each venipuncture. A blinded cardiologist read all 128 ECGs in random order.<sup>43</sup>

There were no significant changes in any of the serum markers. Thirty of the 32 ECG subgroup had normal ECGs for all 4 recordings. One subject had all 4 recordings interpreted as left ventricular hypertrophy and another had occasional sinus pauses in all 4 recordings. The TASER ECD did not affect cardiac or skeletal serum markers or cause serial ECG changes. In other words, no sign of any effect on the heart was found in any of the volunteers.

Another human study, through the University of California at San Diego, found no negative effects on the heart in the 49 volunteers exposed to the TASER devices. 44

The world's expert on fibrillation is Raymond Ideker, MD, PhD who has over 300 indexed manuscripts on the topic and over 1000 total papers and abstracts. He analyzed the TASER X26 output and calculated that it should have a 28:1 safety margin for the typical adult human.<sup>45</sup>

# 6. How Can the ECD Affect the Skeletal Muscles Without Affecting the Heart?

The electrical pulses stimulate the A- $\alpha$  motor nerves which in turn cause the skeletal muscle to contract. The time constant for stimulation of motor neurons from electrodes on the chest of dogs is approximately 0.24 ms, much shorter than the 3.6 ms for cardiac muscle. Other studies give time constants and chronaxies even shorter and nearer to 100  $\mu$ s.

The second reason why the motor neurons are excited by the TASER pulse—while the heart muscle is not—is that the motor neurons are much closer to the electrodes than is the heart. This also explains why the muscles most affected are those nearest to the electrodes of the TASER device. The different electrical resistivities of the various body tissues, and the relative insulating effect of the air-filled lungs, also cause the electric field to be many times smaller in the heart than in the surface tissues near the electrodes. In fact, the amount of current that passes through the heart from electrical pulses delivered to the chest wall is only about 4–10% of the total current delivered through chest electrodes. Most of the current flows around the chest between the 2 electrodes in the intercostal muscles. Electrical current prefers to flow with the grain of a muscle vs. against the grain by a factor of 10:1. Also, the 4–10% value occurs when electrodes are optimally placed to affect the heart. When the electrodes are elsewhere on the

body, as they are in the large majority of cases when the TASER device is used, the percent of applied current that traverses the heart is even less.

Careful computer modeling studies show that very little current goes more than 1 cm below the skin. 51-53

## 7. The TASER ECD is Safer than Some Headache Remedies

Acetaminophen (also sold as paracetamol in the United Kingdom) has caused fatalities in adults with dosages of 15 grams or 30 extra-strength tablets. One in 10 United States poisonings is acetaminophen alone. There are around 250 US deaths annually from acetaminophen. In the UK, it is even more popular and it accounts for every second poisoning.

What is the safety margin for acetaminophen? The bottle labeled safe dose for a single day is 8 tablets. The safety margin for someone taking the daily dose in 1 sitting is thus only 4:1.

To be generous, let us assume that people always followed the timing instructions, which recommend only 2 tablets every 4 hours. (People with bad headaches and other pains do not follow these limits and that is one reason there are so many acetaminophen poisonings.) After 4 hours the last 2 tablets are largely metabolized thus they are counted as — effectively — only 1 in the body. Now add in the 2 new tablets. The body now sees an effective ingestion of 3 tablets, or 1.5 grams. With a lower lethal limit of 15 g, there is now a safety margin of only 10:1.

According to the conservative University of Missouri pig study, the TASER ECD safety margin for a full-sized adult is 30 times, thus a TASER device is indeed far safer than acetaminophen.

How about body size effects? The threshold acetaminophen dose requiring treatment is 150 mg/kg of body weight. Assume a 110 pound young person. Such an individual's body weight is 50 kg, giving a threshold acetaminophen dose of 7.5 grams, or 15 tablets. The safety margin (for the 3 tablets in 4 hours) is 5:1. At this weight, a TASER device has a safety margin of 22:1 by the McDaniel study.

With alcohol, the safety margin of acetaminophen declines precipitously.<sup>54</sup> In fact, there have been deaths in alcoholics using only the recommended dosage.<sup>55</sup>

Regardless of body weight, the TASER device is around 3 to 4 times safer than acetaminophen. Note that this analysis does not include the risk that criminals struck by the TASER device may fall and hit their heads on a hard object, thus risking possible death from the secondary head trauma. This analysis deals only

with death and not injury such as that from a barb hitting the eye or an increased risk of certain cancers associated with acetaminophen.

## 8. Longer TASER ECDs Applications Do Not Affect the Heart.

## Summary

Electricity does not build up in the body like poison. If an electrical current does not electrocute someone in 2–5 seconds, it will not electrocute the person with a longer application. Thus, longer applications have no materially different effect on the heart.

It is well known that VF can be induced by current flowing through the body. The current that is required to cause VF is dependent on the length of time for which the current is applied, and it is well established that the induction threshold decreases for the first few seconds and does not decrease further. In other words, if you are not electrocuted by a certain level of electrical current after 5 seconds, you will not be electrocuted by a 60 second exposure either. If 1 ping-pong ball hit to the head does not kill you, 1,000 probably cannot either.

Both the International Electrotechnical Commission (IEC) and Underwriters Laboratories (UL) regulations recognize that electrocution either happens in the first second (or 2) or does not happen.<sup>56</sup> Currents that will not induce VF in 1 second will not induce VF in 1 minute as shown in Figure 3 taken from Chilbert p 496.

Figure 2 shows that the TASER ECDs are literally off the charts as their currents are too low to fall on the graph.

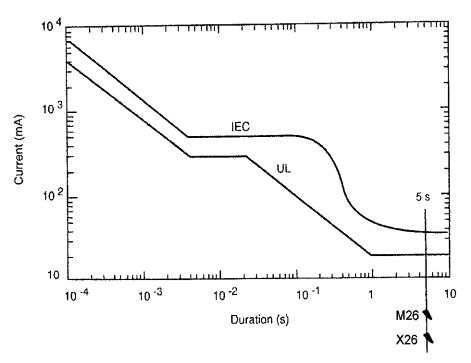


Figure 3. Short applications (less than 1 second) require increasing current levels to induce VF. The lines on this graph represent worst case scenarios with extremely low probabilities of VF and are less than 1/10 the current typically required to electrocute an adult human being.

## **Experimental Results on Long Duration Shocks:**

Animal studies going back to the 1930s show that the risk of inducing VF does not build up (increase) after the first few seconds. Figure 3 (taken from Antoni p 216) summarizes these studies. The time " $t_2$ " is the time at which further applications of current do not increase the risk of inducing VF. These studies have found time  $t_2$  to range from 0.8 to 5 seconds. <sup>1,3-6,24</sup> Based on the animal results above, Beigelmeier and Lee calculated that  $t_2$  ranged from 2-5 seconds for humans due to the lower heartrate. <sup>3,4</sup>

A recent study in smaller pigs (110 lbs) looked at an extreme scenario by burying the TASER probes under the skin and placing a barb over the most sensitive part of the heart. They found no difference between a 5-s and 15-s application in causing ventricular capture (24/25 vs. 28/28, p = NS by Yates-corrected  $\chi^2$ ). (Due to the differences in thoracic geometry this bilateral passage of current through the heart would be impossible in humans with the lung insulation.)

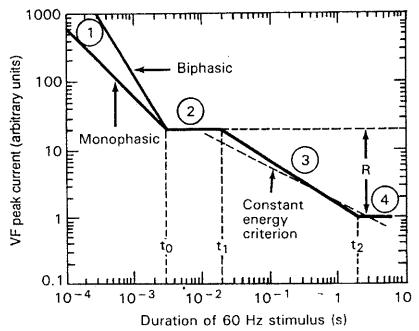


Figure 4. Summary of animal studies going back to 1936 show that at a certain time (t<sub>2</sub>) the risk of electrocution does not increase with longer durations.

One human study found that connecting a 9-volt battery to the inside of the heart could induce VF within 3 seconds in the majority of patients. An internal human study found that the current duration required to fibrillate (96% success rate) with a small steady direct current (DC) was  $3.8 \pm 1.4$  seconds.

The fact that electrocution takes only fractions of a second to a few seconds is also well recognized in pathology.

## 9. Drugs Do Not Make the TASER ECD Dangerous

## Summary

Since illegal drugs are dangerous in so many ways, some uninformed individuals are tempted to assume that they must also be dangerous for electrocution. In fact, the opposite is true. For example cocaine intoxication actually makes a person *harder* to electrocute.

### Cocaine

It is well documented that cocaine has strong effects on the heart such as dramatically raising the heart rate and blood pressure.<sup>57</sup> Cocaine usage also significantly increases the risk of a heart attack <sup>58</sup> and cardiac arrest.<sup>59</sup> Thus, the natural, although incorrect, assumption is that cocaine also makes it easier to electrocute someone. I.e., electrically induce VF. What is surprising — even to some

cardiologists — is that this *natural* assumption is wrong. Most scientific studies have shown that cocaine makes electrocution more difficult — not easier.  $^{60-62}$ 

America's most famous Heart Hospital, the Cleveland Clinic, has recently soundly confirmed these findings using actual TASER X26 waveforms. <sup>41</sup> They found that the safety margin went up significantly and almost doubled for barbs near the heart.

### **Other Stimulants**

With the methamphetamine epidemic there is concern that addicts using this and other illegal stimulants could be at increased risk for electrocution from the TA-SER devices. However, 1 small animal study found no TASER device induced VF even in the presence of various similar stimulating agents.<sup>63</sup>

Cardiac electrophysiologists routinely use intravenous stimulants (such as isoproterenol) to help induce ventricular tachycardias in their electrophysiology lab. Hence, a typical first blush reaction is to assume that stimulants increase the risk from a TASER ECD or external electrical shock in general. However, stimulants — including isoproterenol — tend to actually increase the amount of electrical current required to induce arrhythmias. The same is true with other stimulants such as phenylephrine. 65

The apparent contradiction stems from the fact that electrophysiologists use strong enough pacing pulses (in the electrophysiology lab) directly delivered to a tiny spot on the inside of the heart. So, the decreased sensitivity to electrical stimulation from an adrenergic stimulant is irrelevant in the EP lab — it is trumped by the highly focused and concentrated current delivery. However, this same decreased sensitivity (from adrenergic stimulants) makes externally applied currents much safer. In general, stimulants actually increase the safety margin for externally applied electrical currents.

And, it has long been known that a direct catecholamine (internal stimulants) challenge of epinephrine and norepinephrine appears to only lower the VF threshold for the first 3 to 7 minutes (which is before the arrival of law enforcement to a call for strange or bizarre behavior), after which time the VF threshold actually goes up.<sup>39</sup> Thus, the person, ironically, is probably safer from electrocution than the responding law enforcement officers once they arrive on the scene.

Note that this field situation is far different from someone quickly ingesting a large dosage in front of police officers and then struggling with them. That could be dangerous with some stimulants for the first few minutes as was shown in an absolute worst case situation of low body weight, barb inserted under the skin, barb placed over the heart, and a strong pure stimulant (epinephrine — with no protective effects such as cocaine has) given intravenously.<sup>34</sup>

### Alcohol

A study of intoxicated adults showed that there was no damage from a long (15 second) TASER ECD application.  $^{66}$ 

## Circular Reasoning on Drugs

The typical in-custody death case involves someone with a history of drug abuse as well as possibly an acute toxic level of the same drug of choice. With the overwhelmingly positive field experience with TASER ECDs, no longer does can anyone suggest any danger with a healthy person — without looking silly. However, the same critics point to the few deaths of drug users — in which an ECD was used in the restraint process — and theorize that ECDs are therefore more dangerous with drugs. <sup>67,68</sup> This is completely unscientific for several reasons:

- 1. ECDs are involved in less than 100 of the 20,000 illegal drug deaths annually in the USA.
- 2. Scientific studies show that drugs do not make the ECDs more dangerous. 41,64,65,69
- Crossover data from large and small cities show that implementing ECDs makes suspect injury rates go down and not up.<sup>70</sup>

## 10. Frontal vs. Back TASER Device Applications

There is absolutely zero cardiac effect from TASER ECD applications to the back. This is not to say that there is a risk from using the TASER devices frontally. That has never been scientifically and medically demonstrated in over 700,000 human uses. However, even the most adversarial scientific critics of TASER devices clearly show that there is a zero risk from using these devices on a person's back.

The Nanthakumar porcine study<sup>34</sup> used fairly small swine (110 lbs), much below the typical in-custody-death suspect weight, and were able to induce ventricular fibrillation (VF) in only 1 instance which was highlighted by some for the conclusion of the paper. The salient features of that 1 incidence of VF were: a chemical "trick," or anomaly, to increase the risk of fibrillation, electrodes across the heart and under the skin, and the use of the TASER X26 device. The paper concluded that there was no health risk of cardiac capture or an arrhythmia from electrodes away from the front of the chest. Also the researchers were unable to achieve any sort of cardiac effect with the TASER M26 device and concluded that the higher frequency of its output made it difficult to affect the heart.

Another adversarial porcine study — in which researchers were doing everything possible to electrocute smaller swine with a TASER device —required a modified probe nearly in direct contact with the heart with an open cut in the chest and conductive gel.<sup>30</sup> This research, was from the group of Prof John Webster, who is a highly respected biomedical engineering professor and book author who was

honest enough to admit his *a priori* bias in a media interview that he had personally decided that the TASER devices were occasionally fatal and set out to find out why that was so.<sup>71</sup> He concluded that there was some risk from a barb application directly over the heart in someone so thin that the barb tip came very close to the heart. (As discussed in the Animal Study section, even that risk was exaggerated.)

However, the conclusions of the paper are very helpful here:

Necessary, but not sufficient, conditions for direct electrocution of the heart are 1) dart landing in a small frontal region over the heart suggested by our results, and 2) cardiac arrest of the subject shortly after Taser firing suggested by the literature. Coroners should seek to confirm these conditions before ascribing Tasers as a contributing cause of death. These results suggest that all Taser training should be done on the back, thus avoiding the front of the torso.

#### Conclusion

In summarizing the most relevant features of these adversarial studies it can be concluded that there is zero risk of a TASER device affecting the heart when the application is on the back or when the device is the TASER M26 instead of the TASER X26. In addition the 2 adversarial researchers were only able to induce ventricular fibrillation which is the easiest cardiac arrhythmia to induce and were never able to induce PEA or asystole which are the rhythms usually seen in an in-custody-death.<sup>35</sup>

## 11. The TASER ECD Does Not Interfere with Breathing

Due to the routing of the phrenic nerves it is extremely difficult to electrically induce respiratory paralysis in the human. The phrenic nerve derives from the C3-C5 cervical plexus and the closest passage of phrenic nerves to the skin is just above the clavicle near the sternocleidomastoid muscle. The left and right phrenic nerves travel through the center of the thorax passing just on the margins of the heart on the way to enervate the left and right hemidiaphram muscles. They are surrounded by the highly insulative lungs throughout this passage, thus making them very insensitive to external electrical currents.

This has proved frustrating to researchers seeking to make a respiratory pace-maker following the success of external cardiac pacemakers. Researchers at Purdue University had some success in achieving electrical ventilation in dogs.<sup>72</sup> Probably due to the significantly different thoracic geometry, these results did not carry over to human experiments and the human research attempts have been abandoned.

Even with current forced longitudinally through the whole thorax, the amount of current required to cause temporary respiratory arrest is on the order of the lethal level capable of inducing VF. A classic study found that it could require up to 20-50 mA to cause temporary respiratory arrest in dogs much smaller than humans. This level of current is at least a decade removed from the 2.1 mA delivered by the TASER X26.

In addition, hundreds of videotapes of humans receiving TASER ECD applications demonstrate that respiration is maintained. Subjects are able to talk although the conversations were more filled with profanities than philosophy. In 2005 the United States Air force published a study, using a porcine model, concerned with issues of cardiac safety and rhabdomyolysis. Despite prolonged ECD exposures (90-180 seconds total each) the study found no such cardiac or rhabdomyolysis concerns or problems.

However, the authors made a casual note that the pigs appeared to stop breathing during their 5 second ECD exposure. This result could have been ignored as it was a non-instrumented and off-protocol observation, and if true was probably due to a short-term gasp reflex. In addition, the supine position (on the back) is nonphysiological for swine and the anesthetic used TZ (tiletamine-zolazepam) is known for compromising respiration. However, the researchers intentionally chose TZ as it had minimal effects on creatine phosphokinase which is a marker for muscles damage. Thus, they intentionally were exaggerating breathing effects in order to focus on muscle damage.

The porcine study's relevance to human breathing could also be questioned due to the anatomical differences found to affect electroventilation decades earlier.

A human study was recently performed by staff from several Emergency Departments. Volunteers were instrumented with a breathing monitoring device which showed that the TASER X26 did not interfere with breathing.<sup>77</sup>

The study assessed the breathing capability of human subjects during extended exposures to a TASER ECD. It comprised 52 resting human subjects who underwent breath-by-breath gas exchange monitoring during a 15-second discharge from a TASER X26. The subjects were randomized and placed on an FDA approved pulmonary function measurement device and received either 3 5-second discharges with a one-second break between cycles or had a continuous discharge of 15 seconds applied. Common respiratory parameters were collected before, during and after the exposure. Health histories and demographic information were also collected on the volunteers.

According to this peer-reviewed study, the results indicate that "prolonged CEW (Conducted Energy Weapon also known as ECD) application did not impair respiratory parameters in this population of volunteers. They were unable to detect any respiratory impairment during either prolonged continuous or prolonged in-

termittent CEW exposure in this study population. It does not appear that prolonged CEW exposure causes a decreased tidal volume, hypercapnia, hypoxia, or apnea.

### 12. Excited Delirium

Excited delirium with associated metabolic acidosis<sup>35,40,78-102</sup> is a usually fatal condition with the following typical features:

- 1. Agitation
- 2. Incoherence or disorganized speech
- 3. Clothing removal
- 4. Paranoia or avoidance behavior
- 5. Extreme strength
- 6. Extreme stamina
- 7. Constant motion or hyperactivity
- 8. Imperviousness to pain
- 9. Inappropriate behavior
- 10. History of chronic stimulant abuse or mental illness
- 11. Breaking of shiny objects such as glass and mirrors
- 12. Brief quiet period before collapse

Most simply explained, the person is mentally out of control and exceeds the normal physical limits of the human body until it "burns out." In greater detail, the person's metabolism goes way up, the respiratory system is unable to adequately compensate, the blood becomes acidic, breathing shuts down, the heart shuts down, and the subject is dead.

Clinical results include:

- 1. Acidosis (acidic blood)
- 2. Rhabdomyolysis or kidney damage (if suspect is resuscitated)
- 3. Presenting rhythm of PEA (pulseless electrical activity) or asystole
- 4. Hyperthermia
- 5. Positive Mash<sup>88</sup> test for dopamine transporter assay in brain
- 6. Positive hair test for chronic stimulant abuse 103-107

The term "excited delirium" is fairly modern<sup>93</sup> and this condition has been referred to as Bell's mania, lethal catatonia, <sup>108</sup> and agitated delirum. <sup>92,98-102</sup> Surprisingly, some non-medical critics have questioned its scientific basis since it has no numerical code for insurance billing purposes with the exact words, "excited delirium." Obviously, this is not a condition for which an insured productive member of society makes psychiatric appointments. This is a usually-fatal emergency typically associated with law enforcement involvement.

Similarly, part of the Walter Reed Medical Center scandal surrounds the fact that until recently, there was no numerical code for Traumatic Brain Injury<sup>109-111</sup> so soldiers with this were getting coded as "Organic Psychiatric Disorder". This is just another example of how real conditions exist regardless of whether the insurance billing coding system is keeping up with known medical diagnoses.<sup>112</sup>

As Drs. Farnham and Kennedy so bluntly — and accurately — put it, "... but as psychiatrists we are more interested in the living than the dead and how to prevent the living becoming the dead." 97

## 13. Anti-Police Group Fund-Raising "Studies" are Pure Junk Science.

## **Summary**

Why do some people succumb to sudden unexpected and unforeseeable death while encountering law enforcement officers? We all want answers and we all want to blame something. Often, some people seek to blame a single cause temporal to the person's death, while most often the causation of death is actually multi-factorial and is usually chronic rather than acute. Resisting arrest and consuming illegal drugs, especially over a long period of time, is dangerous and often leads to "excited delirium"-type deaths, which is the most likely explanation for this sad type of death.

The fact that a TASER ECD was used in some attempts to control someone to save their life is not why they died.

This cannot be put any better than it was in the Farnham editorial in the British Medical Journal.<sup>94</sup>

Legal reasoning favours single proximate causes rather than medical conditions, but the intervention most proximate to the time of death is not necessarily the cause of death. Similarly, popular journalism favours controversy and blame rather than balance and exploration.

#### Causation vs. Correlation

Why does the sun come up after the rooster crows? If you think that the rooster's crow caused the sun to come up you have committed the *post hoc ergo propter hoc* logical fallacy. The statement "post hoc ergo propter hoc" is Latin for "after this therefore because of this." You put your coat on before you go out on a winter day. Do you think your coat caused the air to turn cold? That is why some reporters lacking both scientific training and an understanding of statistics may see causations where there are none. 113-115

About 20,000 Americans die annually of drug-related causes according to the Center for Disease Control. A cocaine overdose death is not a peaceful sleepy death like one from heroin – especially if the individual is a chronic abuser. It is violent, disturbing, and with bizarre behavior. It is also ugly, sad and tragic. The methamphetamine epidemic is also causing a rapidly increasing number of stimulant deaths which may top those of cocaine. The end-stage of chronic cocaine or methamphetamine usage is a fairly consistent sequence of events typically referred to "excited delirium." These same symptoms can also be seen in violent struggle deaths triggered by a psychiatric episode. This has been recognized as far back as *before* the American Civil War. The

A decade ago, these in-custody-deaths were blamed on pepper spray. "Pepper Spray: A Magic Bullet under Scrutiny." In that report, the American Civil Liberties Union (ACLU) of Southern California "documented" 7 fatalities after the use of pepper spray in a 9-month period. These deaths have also been blamed on choke-holds 118 and hog-tying or positional asphyxia. 98,119-126 Now the TASER devices are being seen as a simple, "obvious" scapegoat for these tragic deaths.

If a person with normal mental function has excessive exertion, that individual's brain will recognize the signals that the blood is getting acidic and will force the body to slow down. If someone's mental functioning is such that this feedback signal is ignored, the person may struggle until they die. Thus, excited delirium can kill by making the blood so acidic that nothing can function. The typical features of excited delirium are shown in the earlier checklist. The main causes are chronic, illicit stimulant abuse, and the presence of certain mental health conditions.

When someone is dying this horrible death described as excited delirium, they usually exhibit bizarre, unusual, and violent resistive behavior which often attracts attention. Law enforcement personnel are then called and they have to deal with this syndrome for which no good or accepted protocols or treatment have been agreed upon.<sup>95</sup>

Studies of excited delirium deaths show that the majority of the cases have no TASER device involvement.<sup>35</sup> Citizen Down, a law enforcement "watchdog" group, estimates that 1,000 American citizens die in law enforcement custody each year. This is probably an exaggeration as Dr. Jeffrey Ho is able to identify nearly 200 cases per year<sup>37,131</sup> and estimates that the number is about 500. About 58% of the U.S. law enforcement agencies have at least some TASER devices. If the TASER device-carrying officers were to be always called to deal with these violent cases then one would estimate 290 "TASER-related" deaths per year. Consider a much more conservative estimate and take just the 29% of officers, across the country, who carry TASER devices on their belts. That would give a very conservative estimate of 145 "TASER-related" deaths per year. The clear and inescapable conclusion is that the TASER devices are being blamed for far less than their theoretical share of "related" deaths and thus may well be reducing the incidence of in-custody-deaths.